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RELATIVE PULMONIC INSUFFICIENCY.

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In the study of the valvular diseases of the heart, a general agreement among clinicians may be noted in almost every field excepting the one we are to study.

Mackenzie, Dieulafoy, and many other writers do not even mention in their text-books the possibility of relative insufficiency at the pulmonary orifice. Jurgensen was evidently familiar with the murmur, but apparently failed to interpret it correctly. He states,[1] in describing the different types of murmur to be found in mitral stenosis, that "the diastolic murmur is also heard at times in the second left intercostal space, in the region of the pulmonary artery or a little outward from that point. At times the murmur is heard a little more distinctly in that locality than at the apex, especially when a systolic murmur is also present at the latter." It is certainly a most striking circumstance that he describes so closely the murmur we study in the very type of case in which it is most frequently noted. He makes no direct mention elsewhere of the lesion we are considering.

Among the authors at hand who mention the phenomenon definitely are Colbeck, who states that "relative incompetence of the pulmonic valve does sometimes occur, but the lesion is decidedly rare. It . . . may obtain in cases of mitral stenosis."

Satterthwaite states that "temporary insufficiency is doubtless of common occurrence."

Osler[2] says that "both Gibson and Graham Steell have called attention to the possibility of leakage through these valves in cases of great increase of pressure in the pulmonary artery, and to a soft diastolic murmur heard under these circumstances, which Steell calls "the murmur of high pressure in the pulmonary artery."

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[1] Nothnagel's Encyclopedia, Diseases of the Heart.

[2] Eighth edition.

Allyn[3] says that "it is probable that relative pulmonary insufficiency occurs more frequently than we are wont to believe."

Further quotation of authorities would simply emphasize the lack of unanimity of opinion as to the possibility and frequency of the condition.

Gibson's experiments should be quoted in order to show the reasonableness of the diagnosis of relative insufficiency under conditions of high pulmonary arterial pressure. "In the healthy human heart much fluid escaped (from the pulmonary orifice) with a pressure above thirteen inches, a small amount between thirteen and eight inches, and none below that pressure. We have no direct means of estimating the pressure in man. The results of animal experiments give as the mean pressure, 17.6 mm. of mercury in the cat, 12.07 mm. in the rabbit, and 29.6 mm. in the dog (Bentner). Eight inches of water is equal to about 15 mm. of mercury, so that the pressure in the pulmonary artery in man at the height of a vigorous systole of the right ventricle may cause a pressure well above that which first begins to cause insufficiency.[4]

Analogy would lead us to expect to find relative leakage here as at other valves, and it is therefore difficult to undertand the position of certain authors who ignore the existence of the condition, or virtually state that it does not occur.

Personally, I regard relative pulmonary regurgitation as being a not very infrequent complication of advanced mitral stenosis and extensive fibroid phthisis, and as being occasionally found in other conditions in which the pulmonic tension is high.

In "A Clinical Study of Six Hundred Cases of Heart Disease"[5] I found the murmur in question present three times in association with mitral stenosis: once in association with double mitral disease, and once in association with combined aortic and mitral regurgitation. The total number of cases of simple mitral stenosis studied in this series was 35, while 44 cases presented double mitral murmurs. We may safely assume, I believe, that if the signs and symptoms to be mentioned warrant the diagnosis of relative pulmonary insufficiency the affection may probably be found in possibly from 3 per cent. to 5 per cent. of cases of mitral stenosis. My experience before and since the gathering of the statistics quoted (between 1903 and 1909) confirms me in this opinion.

The diagnosis rests upon the association of the following findings:

1. Some definite cause for increased pulmonary tension, notably mitral stenosis or fibroid phthisis in an advanced state.
2. The presence of increased precordial area of dulness, especially suggesting dilatation of the right ventricle.
3. The presence of a soft diastolic murmur heard at the region of the pulmonary valves, and transmitted downward from the

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[3] Amer. Jour. Med. Sci., October, 1913, No. 4, cxlvii, 54.

[4] Quoted from Osler's System of Medicine.

[5] Med. Record, November, 13, 1909.

second or third left intercostal space along the left side of the sternum. The murmur is exactly of the type found in aortic regurgitation, excepting that it is generally more soft and blowing in character. The most striking single feature is the inconstancy of the murmur. After being present for days or weeks, with the regaining of a better condition of compensation, the murmur disappears, to reappear with the next breakdown. It is absolutely essential to the diagnosis that the aortic valve sounds be clear and that no feature of aortic regurgitation be present. Fortunately, the insufficient filling of the left ventricle in mitral stenosis oftentimes renders the aortic second sound so notably feeble that the pulmonary signs are prominent by contrast.

4. Absence of thrill. This is not infrequent in the organic lesion, but is, I believe, never found in relative insufficiency, and should therefore count against such a diagnosis. The presence of a systolic murmur at this orifice would militate against the diagnosis of relative leakage and be in favor of the organic type.

5. The murmur may be increased in intensity by expiration, especially with the closed glottis, the increased intrapulmonary pressure being the active agency in bringing about the exaggeration of the murmur. A similar increase may be noted after exertion.

As is to be expected, the pulmonary second sound is commonly enfeebled by the failure of close apposition of the pulmonic leaflets.

Of the considerable number of cases which I have seen I shall quote but two.

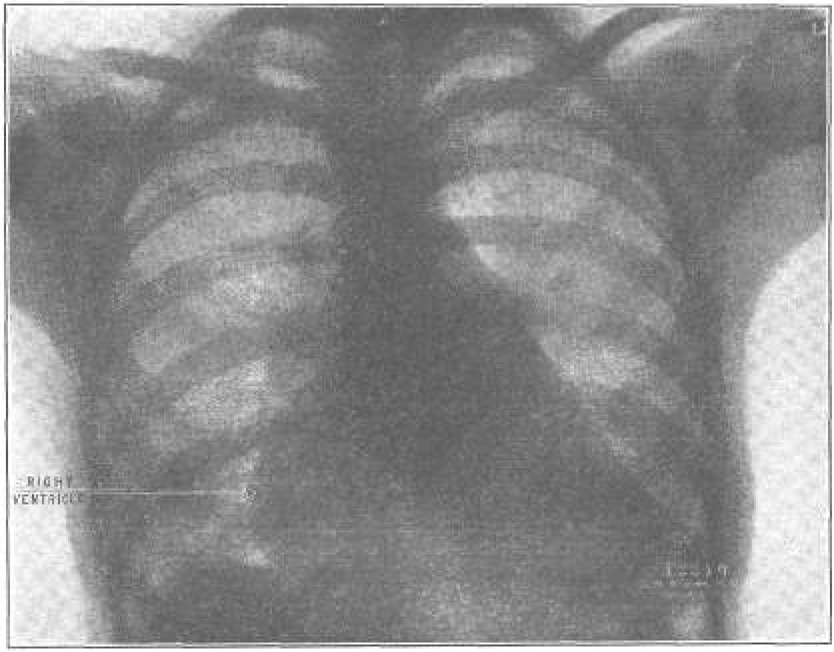
Case I.—Male, aged twenty-seven years, complained of dyspnea and cough, the former of extreme severity. Cyanosis was present at various times while under observation. Edema finally appeared. The heart was much increased in area, the apex-beat being found 12 cm. to the left of the central line, with marked presystolic murmur and thrill. A soft diastolic murmur was found extending downward from the third left interspace. This disappeared under rest in bed and the administration of digitalis. There were none of the features of aortic regurgitation.

The patient later passed through an attack of acute pneumonia, the murmur reappearing. Recovery ensued, this being one of the few cases I have known of recovery from acute pneumonia in advanced mitral stenosis. Within a year the patient died of cardiac incompensation. The postmortem examination showed a marked "buttonhole" mitral valve, with hypertrophy and extreme dilatation of the heart. The aortic, tricuspid, and pulmonic valves were normal.

This case was shown at a meeting of the Denver County Medical Society, and the presence of a murmur of the character described was verified by many of those present.

Case II.—A female, aged thirty-three years, had suffered twice from acute rheumatism. She presented at the single examination which I had the opportunity of making, enormous dilatation of the heart, the transverse diameter of the precordial flatness being 20 cm. The great increase in this direction is well shown in the accompanying skiagram, for which I am indebted to Dr. G. H. Stover. A loud presystolic murmur was present at the apex, with presystolic thrill. At the pulmonic area there was heard a diastolic murmur such as we have described, transmitted downward, and increased in intensity by exertion.

The aortic second sound was notably feeble, and no signs of aortic insufficiency existed. In the skiagram may be noted at *a* the marked dilatation of the right ventricle, and the general increase in area in a transverse direction as mentioned above (60 per cent according to Dr. Stover's measurements). In the absence of post-mortem proof in this case we can, of course, make only a presumptive diagnosis of relative insufficiency.



Skiagram of Case II, showing increased transverse diameter of the dilated heart.

In view of the evidence of mitral obstruction, of the pulmonic diastolic murmur, increased in intensity upon exertion, absence of thrill, absence of signs of aortic disease, feeble aortic second sound, the known rarity of organic pulmonary regurgitation and the entire reasonableness of a diagnosis of relative leakage at the valve mentioned under conditions of marked increase in the tension of the pulmonary circulation, the diagnosis in this and similar cases, in my opinion, rests upon as secure a basis as that of the other valvular diseases of the heart.